In the Specification:

Paragraph numbers indicated herein below reflect those listed in published United States Patent Application No.2004-0166517, which corresponds to the instant application. Accordingly, please amend the Specification as follows:

[0004] Breast cancer is the most frequently diagnosed non-skin cancer among women in the United States. It is second only to lung cancer in cancer-related deaths. In the UK, breast cancer is by far the commonest cancer in women, with 34,600 new cases in 1998 (Cancer Research Campaign, ~~http://www.crc.org.uk,~~ UK, 2000). Ninety-nine percent of breast cancers occur in women. The risk of developing breast cancer steadily increases with age; the lifetime risk of developing breast cancer is estimated to be 1 in 8 for women in the US. The annual cost of breast cancer treatment in the United States is approximately $10 billion (Fuqua, et. al. 2000, American Association for Cancer Research, ~~www.aacr.org,~~ USA). Breast cancer incidence has been rising over the past five decades, but recently it has reached a plateau. This may reflect a period of earlier detection of breast cancers by mammography. A number of established factors can increase a woman's risk of having the disease. These include older age, history of prior breast cancer, significant radiation exposure, strong family history of breast cancer, upper socioeconomic class, nulliparity, early menarche, late menopause, or age at first pregnancy greater than 30 years. Prolonged use of oral contraceptives earlier in life appears to increase risk slightly. Prolonged postmenopausal oestrogen replacement increases the risk 20 to 40%. It has been speculated that a decrease in the age at menarche, changing birth patterns, or a rise in the use of exogenous estrogens has contributed to the increase in breast cancer incidence (Fuqua, et. al. 2000, American Association for Cancer Research, ~~www.aacr.org,~~ USA).

[0006] Breast cancer is a heterogeneous disease. Although female hormones play a significant role in driving the origin and evolution of many breast tumours, there are a number of other recognised and unknown factors involved. Perturbations in oncogenes identified include amplification of the HER-2 and the epidermal growth factor receptor

genes, and over-expression of cyclin D1. Over-expression of these oncogenes has been associated with a significantly poorer prognosis. Similarly, genetic alterations or the loss of tumour suppressor genes, such as the p53 gene, have been well documented in breast cancer and are also associated with a poorer prognosis. Researchers have identified two genes, called BRCA1 and BRCA2, which are predictive of premenopausal familial breast cancer. Genetic risk assessment is now possible, which may enhance the identification of candidates for chemoprevention trials (Fuqua, et. al. 2000, American Association for Cancer Research, ~~www.aacr.org,~~ USA).

[0008] Early diagnosis of breast cancer is vital to secure the most favourable outcome for treatment. Many countries with advanced healthcare systems have instituted screening programs for breast cancer. This typically takes the form of regular x-ray of the breast (mammography) during the 50-60 year old age interval where greatest benefit for this intervention has been shown. Some authorities have advocated the extension of such programs beyond 60 and to the 40-49 age group. Health authorities in many countries have also promoted the importance of regular breast self-examination by women. Abnormalities detected during these screening procedures and cases presenting as symptomatic would typically be confirmed by aspiration cytology, core needle biopsy with a stereotactic or ultrasound technique for non-palpable lesions, or incisional or excisional biopsy. At the same time other information relevant to treatment options and prognosis, such as oestrogen (ER) and progesterone receptor (PR) status would typically be determined (National Cancer Institute, USA, 2000, Breast Cancer PDQ~~,~~ ~~www.nci.org~~).

[0014] The Diastrophic Dysplasia (DTD) gene was cloned in 1994 (Hastbacka J, et al. Cell 1994, 78(6): 1073-87). Its sequence is stored in the Swiss-Prot database (held by the Swiss Institute of Bioinformatics (SIB) ~~and available at http://www.expasy.ch~~ under the accession number P50443 and encodes a putative 12-membrane sulfate transporter of 739 amino acids (FIG. 1, SEQ ID NO: 1). It is located on chromosome 5q31-q34.

[0209] FIG. 1: shows the amino acid sequence (SEQ ID NO: 1) and nucleic acid sequence (SEQ ID NO: 2) of DTD, the sequence is stored in the Swiss-Prot database (held by the Swiss Institute of Bioinformatics (SIB) and available at ~~http://www.expasy.ch/~~) under the accession number P50443.

[0215] The gene for DTD has been localised to chromosome 5q31-q34 (Hastbacka J, et al. Genomics 1991;11:968-73). A Blast search ~~(http://www.ncbi.nlm.nih.gov/BLAST/)~~ with the DTD gene against High-Throughput Genome Sequences (HTGS) returns the GenBank clone: AC011406. The DTD sequence was amplified using PCR from a colon cDNA library using the following primer pair:

1 Sense: 5' aggaagctgaaccatctatctc 3' (SEQ ID NO: 3) Antisense: 5' actgggaaatgttggacacttg 3' (SEQ ID NO: 4)

[0216] Alignment of the nucleotide sequence from FIG. 1 (SEQ ID NO: 2) and part of the genomic clone AC011406 demonstrated the presence of 2 exons, with the boundary between each exon being around bp724-bp729 in the sequence shown in FIG. 1 (SEQ ID NO: 2). Dotlet, ~~accessible at http://www.isrec.isb-sib.ch/java/dotlet/Dotlet.html,~~ was used to align the two sequences.